IN THE CLAIMS

Please amend the claims as follows:

1. (Currently amended) A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ~~wild-type~~ ATF2, wherein the inhibitory N-terminal fragment of ATF2 comprises amino acid residues selected from the group consisting of:

   i. from about amino acid residue 1 to about amino acid residue 115 of ATF-2;

   ii. from about amino acid residue 50 to about amino acid residue 100 (Peptide II) of ATF-2 [[.]];

   iii. from about amino acid residue 45 to about amino acid residue 75 of ATF-2;

   iv. from about amino acid residue 45 to about amino acid residue 100 of ATF-2; and

   v. from about amino acid residue 50 to about amino acid residue 75 of ATF2.

2. (Canceled)

3. (Canceled)

4. (Currently amended) ~~The method of claim 1~~ A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of ~~amino acid residues from~~ about amino acid residue 50 of ATF2 to about amino acid residue 75 of ATF2.

5. (Canceled)

6. (Canceled)

7. (Canceled)

8. (Original) The method of claim 1 wherein the tumor cell is a melanoma tumor cell.

9. (Original) The method of claim 1, wherein the tumor cell is a breast cancer tumor cell.

10. (Original) The method of claim 1, further comprising treating the tumor cell with a chemotherapeutic agent.

11. (Original) The method of claim 10, wherein the chemotherapeutic agent is selected from the group consisting of a p38 inhibitor, UCN-01, NCS, anisomycin, LY294002, PD98059, AG490, and SB203580.

12. (Previously presented) The method of claim 1, further comprising treating the tumor cell with radiation, wherein the inhibitory N-terminal fragment of ATF2 sensitizes the tumor cell to the radiation.

13. (Previously presented) A polypeptide comprising an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of amino acid residues from about residue 50 to about residue 100.

14. (Canceled)

15. (Original) The polypeptide of claim 13, further comprising a translocation peptide sequence.

16. (Withdrawn) A nucleic acid encoding a polypeptide comprising an inhibitory ATF2 N-terminal fragment, which N-terminal fragment comprises a sequence from about amino acid residue 50 to about amino acid residue 75 of ATF2.

17. (Withdrawn) The nucleic acid of claim 16 encoding a polypeptide wherein the N-terminal fragment comprises from about amino acid residue 45 to about amino acid residue 100 of ATF2.

18. (Withdrawn) An expression vector comprising the nucleic acid of claim 16 operably associated with an expression control sequence.

19. (Withdrawn) The expression vector of claim 18, wherein the expression control sequence provides for expression in a tumor cell.

20. (Original) A pharmaceutical composition comprising the polypeptide of claim 13 and a pharmaceutically acceptable carrier or excipient.

21. (Original) A pharmaceutical composition comprising the polypeptide of claim 15 and a pharmaceutically acceptable carrier or excipient.

22. (Withdrawn) A pharmaceutical composition comprising the expression vector of claim 18 and a pharmaceutically acceptable carrier or excipient.

23. (Previously presented) A method of treating a tumor in a subject, which method comprises administering therapeutically effective amount of the pharmaceutical composition of claims 20 or 21, to the subject.

24. (Original) The method of claim 23 wherein the tumor is a melanoma tumor.

25. (Original) The method of claim 23, wherein the tumor is a breast cancer tumor.

26. (Original) The method of claim 23, further comprising treating the tumor with a chemotherapeutic agent.

27. (Original) The method of claim 26, wherein the chemotherapeutic agent is a p38 inhibitor.

28. (Original) The method of claim 26, wherein the chemotherapeutic agent is selected from the group consisting of UCN-01, NCS, anisomycin, LY294002, PD98059, AG490, and SB203580.

29. (Previously presented) The method of claim 23, further comprising treating the tumor with radiation, wherein the inhibitory N-terminal fragment of ATF2 sensitizes the tumor cell to killing by the radiation.

30. (Withdrawn) A method for identifying a compound that modulates ATF2 activity, which method comprises determining the level of expression of a reporter gene in a cell comprising the reporter gene operatively associated with an ATF2-regulated expression control sequence contacted with a compound under conditions in which ATF2 would induce expression of the reporter gene in the absence of the compound, and comparing the level of expression of the reporter gene in the presence of the compound to the level of expression in the absence of the compound, wherein a difference in the level of expression of the reporter gene indicates that the compound modulates ATF2 activity.

31. (Withdrawn) The method of claim 30, wherein the level of reporter gene expression in the presence of the compound is less than in the absence of the compound, wherein the compound inhibits ATF2 activity.

32. (Withdrawn) The method according to claim 31, wherein the compound is a polypeptide.

33. (Canceled)

34. (Canceled)

35. (Previously presented) The method of claim 1, wherein contacting the cell with the inhibitory N-terminal fragment increases the activity of a c-jun family member in the cell, as compared to the activity of the c-jun family member in a tumor cell not contacted by the fragment.

36. (Previously presented) The method of claim 35, wherein the c-jun family member is jun kinase (JNK).

37. (Previously presented) The method of claim 35 wherein the tumor cell is a melanoma tumor cell.

38. (Previously presented) The method of claim 35, wherein the tumor cell is a breast cancer tumor cell.

39. (Previously presented) The method of claim 35, further comprising treating the tumor cell with a chemotherapeutic agent.

40. (Previously presented) The method of claim 39, wherein the chemotherapeutic agent is selected from the group consisting of a p38 inhibitor, UCN-01, NCS, anisomycin, LY294002, PD98059, AG490, and SB203580.

41. (Previously presented) The method of claim 40, wherein the chemotherapeutic agent is a p38 inhibitor.

42. (Previously presented) The method of claim 41, wherein the tumor cell is a late stage melanoma cell.

43. (Previously presented) The method of claim 35, further comprising treating the tumor cell with radiation.

44. (Canceled)

45. (Canceled)

46. (Canceled)

47. (Canceled)

48. (New) A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of about amino acid residue 1 of ATF2 to about amino acid residue 115 of ATF2.

49. (New) A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of about amino acid residue 50 of ATF2 to about amino acid residue 100 of ATF2.

50. (New) A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of about amino acid residue 45 of ATF2 to about amino acid residue 75 of ATF2.

51. (New) A method of inhibiting growth of a tumor cell, which method comprises inhibiting transcriptional activity of ATF2 by contacting the cell with an inhibitory N-terminal fragment of ATF2, wherein the inhibitory N-terminal fragment of ATF2 consists essentially of about amino acid residue 45 of ATF2 to about amino acid residue 100 of ATF2.